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Clinical results of common strategies used to revise infrainguinal vein grafts

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Purpose: Patients who have failing infrainguinal bypass grafts or failed grafts reopened with lytic therapy represent a group at high risk of subsequent failure. Previous studies suggest that vein patch angioplasty and jump grafting may be less durable than interposition grafting as a method of correcting graft lesions. Our objective was to assess the value of various technical strategies for graft revision in a series of autogenous infrainguinal bypass grafts and to assess how these variables might affect cumulative graft patency (CGP) rates.

Methods: We retrospectively reviewed the clinical course, anatomic sites of revision, and type of revision performed on 67 grafts in 58 patients who underwent at least one revision from 1991 to 1995. Results were assessed with regression analysis and Kaplan-Meier estimates of CGP rates ($p < 0.05$ was considered significant).

Results: Sixty-seven vein grafts underwent revision of 112 anatomical sites in 95 operations. Forty-nine of 67 grafts were single-segment greater saphenous vein grafts and 18 were composite (>1 segment) grafts, with an overall 5-year CGP rate of 72%. No difference was observed between the 4-year CGP rate in grafts with hemodynamically significant distal anastomotic stenoses repaired primarily with jump grafts (n = 20, 71% CGP rate) and those with stenoses found only in the graft body (n = 41, 89% CGP rate). Vein patch angioplasty was used primarily, but not exclusively, for focal graft body stenoses (n = 35), whereas interposition grafts (n = 11) were reserved for more diffuse strictures; no significant difference in 3-year CGP rates was observed (94% and 73%, respectively).

Conclusion: Using an appropriate revision strategy that favors vein patch angioplasty for graft body lesions and jump grafts for distal anastomotic lesions, acceptable assisted patency rates can be achieved in grafts that are at risk for repeated failure. (J Vasc Surg 1996;24:909-19.)

The concept of the failing autogenous infrainguinal bypass graft is now well accepted by vascular surgeons, whose task it is to identify the stenotic but still patent graft and revise it before thrombosis occurs. Thrombosis, even with early thrombectomy or lyric therapy followed by revision, portends reduced graft longevity.[1-3] Once the failing vein graft is identified and well characterized with respect to clinical, hemodynamic, and anatomic data, the vascular surgeon must choose the appropriate operation to maintain graft patency. Although there have been numerous reports that have documented that vein graft revision enhances the primary patency rates of both in situ and reversed saphenous bypass grafts,[4-9] specific strategies that address anatomic disease and technical management of graft failure have been limited. The purpose of this study was to review our experience with a cohort of 58 patients who underwent at least one revision of a failing or failed but salvageable infrainguinal autogenous lower extremity bypass graft. This study is designed to characterize the anatomic causes of graft failure and outline our experi-

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Table I. Clinical presentation and noninvasive laboratory evaluation of 67 lower extremity vein grafts before 95 surgical revisions (67 primary and 28 secondary revisions)

Symptoms	*No. of revisions*	*ABI*	*Peak systolic flow velocity*	
			Midgraft	*Stenosis*
Asymptomatic	24	0.8 ± 0.04	55 ± 5	371 ± 29
Claudication	34	0.8 ± 0.03	61 ± 6	314 ± 17
Rest pain or tissue loss	37	0.4 ± 0.03*	45 ± 5	348 ± 42

*$p < 0.02$.

ence with several different revision strategies for graft salvage.

METHODS

During the 5-year period from January 1991 to December 1995, 58 patients underwent surgery for a failing or failed (opened by thrombectomy or lytic therapy) autogenous infrainguinal bypass grafts at New England Medical Center. Sixty-seven grafts were revised at least once, whereas 23 were revised on two or more occasions, for a total of 95 revisions. Patent grafts with impending failure (56 of 67 grafts) were studied angiographically when graft deterioration was identified by clinical or noninvasive laboratory evaluation. Clinical deterioration was suspected if a history of symptom progression was reported or if physical examination revealed a change in the graft or distal pulse quality. Graft deterioration in both symptomatic and asymptomatic patients was further evaluated with segmental arterial Doppler pressure measurements and pulse volume recordings before and after exercise. An decrease in ankle-brachial index more than 0.15 was considered significant enough to warrant further graft evaluation. Surveillance duplex ultrasonographic scans (ATL, HDI System, Bothell, Wash., L10-5 MHz probe) of the entire graft were performed when graft deterioration was suspected or as part of routine postoperative surveillance, unless the clinical situation or an abnormally low ankle-brachial index dictated arteriographic evaluation. Peak systolic flow velocity was measured at multiple points on the graft, focusing on suspected stenotic areas. A peak systolic flow velocity greater than 300 cm/sec in an asymptomatic limb represented a graft-threatening stenosis and prompted an arteriographic scan in most instances, unless the patient was medically unfit for surgery or autogenous tissue was unavailable for graft revision. Because of individual surgeon preference for the graft surveillance method as well as variations in patient compliance, 63% of grafts underwent duplex scans before the first revision, whereas 75% of grafts that were revised more than once were scanned before surgery. Thirty-one percent of grafts were studied with pulse volume recordings and segmental arterial pressure measurements alone before the arteriographic scan and revision.

Of the 67 original grafts that underwent revision, 49 were single-segment greater saphenous vein grafts, including 31 in situ grafts, 15 nonreversed translocated grafts, and three reversed grafts. Eighteen were composite grafts (more than one segment) spliced with arm vein only (10), greater saphenous vein (5), lesser saphenous vein (1), or some combination of these (2). The initial bypass graft was to the above-knee popliteal artery in five limbs, the below-knee popliteal artery in 16 limbs, and to the tibial vessels in 46 limbs.

Graft failure in the occluded bypass grafts that was detected at the time of the first revision (11 of 67 grafts) was heralded by a return to, or worsening of, prebypass symptoms and arterial pressures, as well as a loss of graft pulse. Graft thrombosis was confirmed either by Duplex scan, by a standard angiogram, or both, with expedient selective use of lytic therapy or prompt thrombectomy to open the graft and identify an anatomic reason for failure. Data were collected by reviewing the hospital records, including noninvasive laboratory studies, angiograms and reports, and operative notes, as well as by personal interviews during follow-up (reviewed in Table I). Details of the gross pathologic character of the lesions that were responsible for impending graft failure were gleaned primarily from operative notes and were sufficient for characterization of these lesions in 81 of 95 revisions.

The secondary operations that were used to correct 112 anatomic lesions in 95 operations included vein patch angioplasty (VPA; 60) for focal lesions (<2 cm), vein interposition grafts (graft, 15), vein jump grafts (25), polytetrafluoroethylene patch angioplasty (6), balloon angioplasty (6), or some combination of these (14). A jump graft is an autogenous extension of the bypass from the normal vein graft proximal to a stenosis to nondiseased distal native artery.

The mean follow-up period for this group of 67 grafts was 39 months from the original operation and 25 months from the first revision. Cumulative graft patency (CGP) rates, patient survival rates, and limb salvage rates were calculated using life-table analy-

Table II. Anatomic location of 112 vein graft stenoses identified before revision and method used to correct them

		Repair				
Site of primary revision	*No. of lesions*	*VPA*	*IP graft*	*Jump graft*	*PTFE patch*	*PTA*
Proximal anastomosis	5	1			3	1
Distal anastomosis	15	5		10		
Midgraft	49	37	8	3		1
Native artery	6	2		2		2
Site of secondary revision						
Proximal anastomosis	5		1	1	3	
Distal anastomosis	5	2		3		
Midgraft	10	5	4	1		
Venovenostomy	4	4				
Vein patch	6	3	2	1		
Native artery	7	1		4		2
Total	112	60	15	25	6	6

IP graft, Interposition graft; *PTFE,* polytetrafluoroethylene patch angioplasty; *PTA,* percutaneous transluminal angioplasty.

sis.[10] Limb salvage rates were expressed in terms of clinically functional limbs. The statistical analysis was performed with Stat View 4.5 statistical software (Abacus Concepts, Berkeley, Calif.). Kaplan-Meier life-table estimates were constructed with differences between groups assessed using a proportional hazards regression analysis. Comparisons of group characteristics were performed using analysis of variance with Newman-Keuls post hoc testing (p value less than 0.05 was considered significant).

RESULTS

The anatomic location of 112 infrainguinal bypass graft or native arterial lesions that underwent 95 revisions and the type of revision performed are reviewed in Table II. The majority of the revisions were performed for lesions that were confined to the conduit, excluding the anastomoses (69 of 112, 62%). Forty-nine of 69 midgraft conduit lesions were repaired at the first revision, whereas 20 were addressed at subsequent secondary revisions. VPA was the most commonly performed operation; it was used to correct 57 vein graft stenoses and three native artery lesions. Seven of the 60 VPAs were performed on lesions at the distal anastomosis, three of which were at the ankle level. Twenty-seven percent of the lesions corrected in 23 re-revisions were at sites of previous VPA or venovenostomy, whereas 46% were new secondary lesions that did not involve an anastomotic site.

Gross pathologic lesions that were responsible for the failing graft and the interval to revision are depicted in Fig. 1. Although some grafts at the time of revision had more than one site of stenosis, this graph reflects the predominant lesion that was responsible for impending failure or thrombosis. Intimal hyperplasia was identified as the primary cause of graft stenosis in 63 intrinsic stenoses, including 23 proximal and distal anastomotic lesions and 40 midgraft lesions that developed de novo or at valve sites or sites of previous venovenostomy. All ten proximal anastomotic stenoses occurred in nonreversed saphenous vein grafts. Progression of atherosclerosis contributed to graft failure, with 18 treatable lesions identified. Thirteen stenoses were located in the native arterial circulation; five were attributed to late atherosclerotic progression in the vein graft itself. The interval to revision in grafts that failed because of native artery progression of atherosclerosis was significantly greater than in grafts that failed for any other reason ($p < 0.05$). Seven of the 67 grafts had two or more intrinsic stenoses in separate areas of the midgraft (excluding the anastomoses). Eleven of the 67 grafts were occluded at the time of first revision, and two additional occlusions occurred after the first revision. Nine of these 13 occluded grafts were opened with urokinase, and the remaining four grafts underwent thrombectomy in the perioperative period. Six of these 13 grafts have remained patent at the last follow-up, with two grafts remaining open at 45 months.

The overall CGP rate by life-table analysis (Fig. 2 and Table III) for the 67 grafts that underwent at least one revision was 72% at 5 years. The CGP rates of grafts that were originally constructed as multisegment composite grafts was 56%, compared with a 95% rate for single-segment greater saphenous vein grafts at 30 months (Fig. 2 and Table IV; $p < 0.05$).

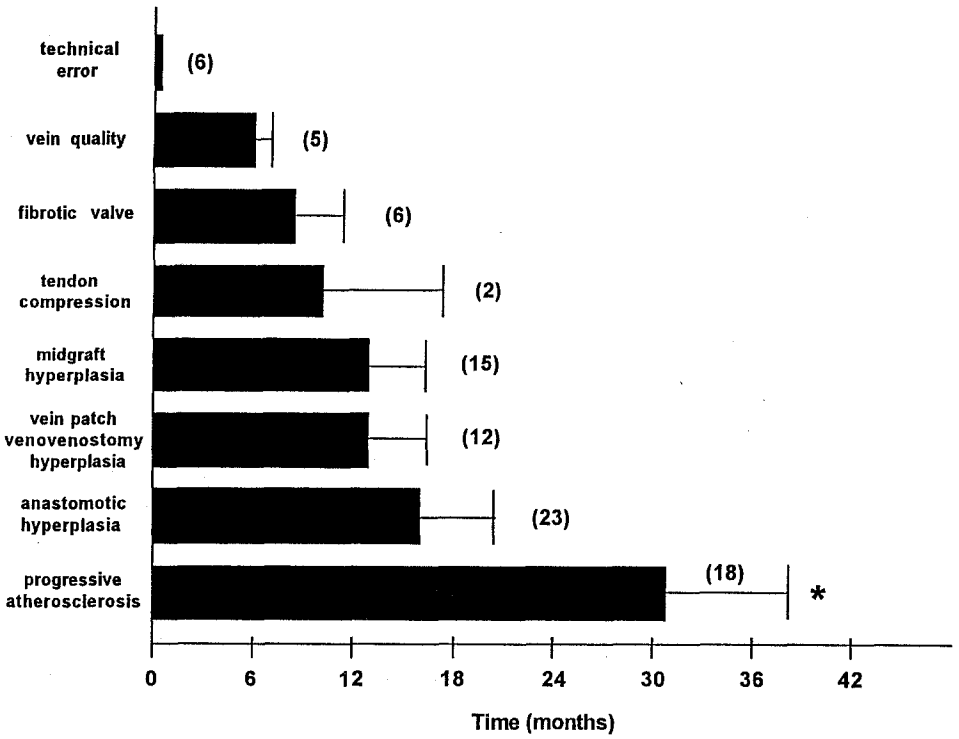

Fig. 1. Comparison of causes of infrainguinal autogenous bypass graft failure leading to revision and time interval from original bypass to discovery of graft failure. Vein graft failure caused by progression of atherosclerosis occurred later than failure associated with other causes. $*p < 0.05$ by analysis of variance.

The CGP rate of grafts that required correction of a distal anastomotic stenosis at some point during the life of the graft was 71% at 4 years, whereas grafts that underwent graft body revision alone had a 89% CGP rate at 4 years (Fig. 3 and Table V; p = NS). Graft body stenoses that were repaired with one or more VPA procedures had a 3-year CGP rate of 94%, whereas those repaired with interposition grafts had a CGP rate of 73% at 3 years, with no significant difference in patency rates noted. The 4-year CGP rate for grafts that were revised with a distal jump graft was 83%. The 3-year secondary assisted patency rate for the entire group of 67 grafts was 70% (data not shown).

The perioperative morbidity rate for the 95 vein graft revisions was 9%. Wound complications developed in three patients, and there were three minor bleeding complications that were related to perioperative heparin use. Two patients had myocardial infarction without hemodynamic compromise. A graft infection with hemorrhage from a dehisced proximal anastomosis developed in one patient and resulted in graft failure and amputation. The overall limb salvage rate for patients who underwent revision procedures was 83% at 5 years, and the 5-year survival rate after the first revision was 72%.

DISCUSSION

Since the introduction of the autologous saphenous vein bypass for lower extremity ischemia by Kunlin in 1948, a great deal has been learned about the natural history of the arterialized venous conduit. In 1973 Szilagyi et al.[11] characterized six important morphologic alterations that are observed angiographically, as well as in postmortem and surgical pathologic vein graft specimens. In this landmark study, they found that intimal thickening (neointimal hyperplasia) was the most common abnormality contributing to graft failure, which he described as "wavy narrowing" that diffusely involved extensive portions of grafts. He also described the fibrotic valve and traumatic stenosis, which we now know are both also related to neointimal hyperplasia in response to local graft flow disturbance or intimal trauma.[12] Atherosclerotic degeneration, typical of the native artery, that contributed to graft failure was observed as a late

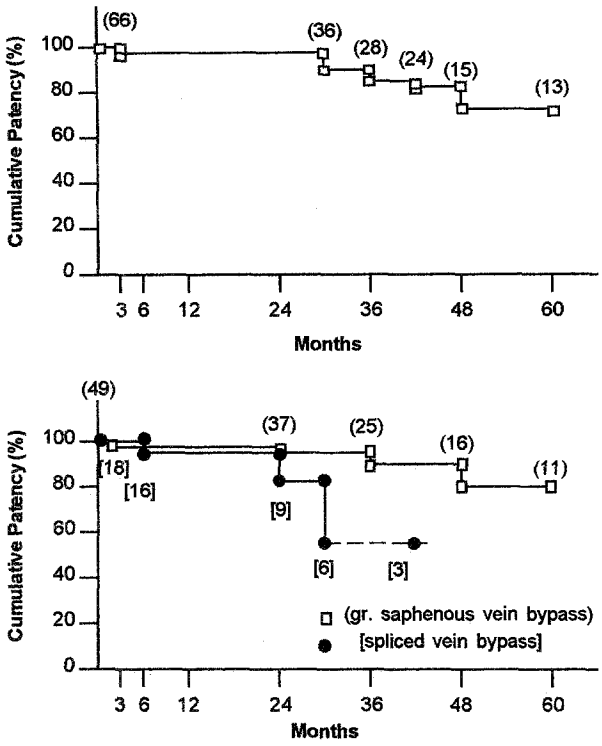

Fig. 2. Cumulative primary assisted patency data of 67 infrainguinal autogenous bypass grafts that underwent at least one revision *(upper panel)*. CGP rates of 49 greater saphenous vein versus multiple vein segment lower extremity bypass grafts were 95% and 56% at 30 months ($p < 0.05$) *(lower panel)*.

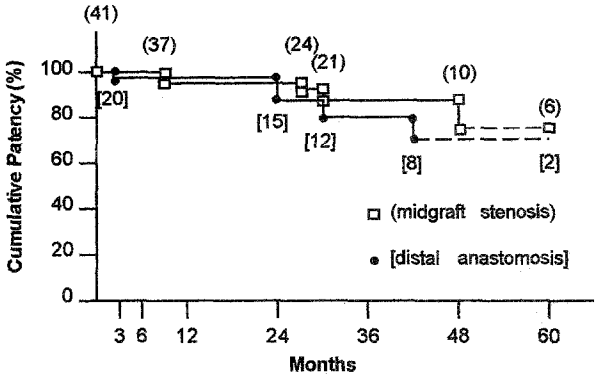

Fig. 3. Comparison of 4-year cumulative patency data of grafts revised for midgraft versus distal anastomotic stenosis regardless of method used for revision.

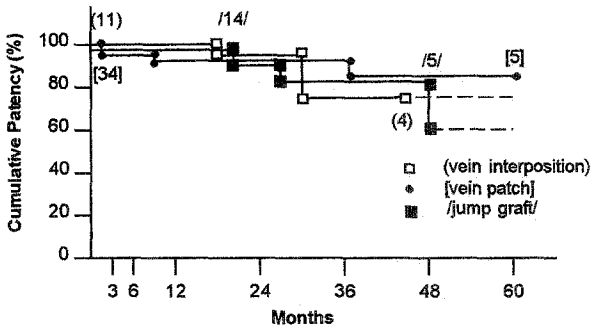

Fig. 4. Life-table analysis of failing or failed lower extremity vein grafts that underwent revision with either VPA, interposition graft, or jump graft.

finding in 8% of grafts, but interestingly, it was observed histologically in 80% of specimens that were taken from grafts that had been in place 2 years or longer. The other two less common entities were suture stenosis at vein branches and late aneurysmal degeneration of the vein. Nowhere in Szilagyi's study, however, is there mention of anastomotic stenosis as a cause of vein graft failure.

In 1981 Whittemore et al.[1] reported 109 femoropopliteal vein graft failures in which vein graft stenosis was the most common cause of failure. In that report the highest CGP rate was achieved in the grafts that had short-segment stenoses repaired with VPA before graft thrombosis: 86% versus 19% 5-year CGP rates in grafts that required thrombectomy. Subsequent reports have shown that VPA performed for focal stenoses in patent grafts is superior to percutaneous transluminal angioplasty.[2,5] In 1992 Berkowitz et al.[6] reported anatomic data in a series of reversed saphenous vein graft stenoses in which the majority of lesions (40%) were just distal to the proximal anastomosis hood and 29% were juxtaanastomotic, evenly split between proximal and distal. The propensity for proximal midgraft lesions in their series may have been a result of the natural tapering of the saphenous vein proximally when placed in the reversed position. Sanchez et al.[8] reported their observations with 72 failing vein grafts in a series of 120 infrainguinal reconstructions in which they observed a strikingly low 9% juxtaanastomotic stenosis incidence. Their conclusion was that perhaps undetected anastomotic stenosis favors enhanced graft thrombosis, which accounts for a disproportionate loss of grafts that have these lesions.

A report by Donaldson et al.[7] detailed the causes of primary failure of 85 in situ bypass grafts and found that 63% of graft thromboses were caused by intrinsic graft lesions, 27% by juxtaanastomotic lesions, and

Table III. Cumulative patency data for greater saphenous and composite vein bypass grafts

Interval (mo)	*No. of grafts at risk*	*No. of failed grafts*	*No. of grafts withdrawn*	*Interval patency rate (%)*	*Cumulative patency rate (%)*	*Standard error (%)*
0-3	67	1	0	98	98	1.5
3-6	63	0	3	100	98	1.5
6-12	58	1	4	99	97	2.2
12-18	55	0	7	100	97	2.2
18-24	46	0	7	100	97	2.2
24-30	36	2	5	95	92	3.8
30-36	28	2	6	95	87	5.0
36-42	24	1	3	97	84	5.9
42-48	15	0	9	100	84	5.9
48-54	12	2	1	88	72	9.0
54-60	11	0	1	100	72	9.0

Table IV, A. Cumulative graft patency data for greater saphenous bypass grafts undergoing revision

Interval (mo)	*No. of grafts at risk*	*No. of failed grafts*	*No. of grafts withdrawn*	*Interval patency (%)*	*Cumulative patency (%)*	*Standard error (%)*
0-3	49	1	0	98	98	2.0
3-6	47	0	1	100	98	2.0
6-12	44	0	3	100	98	2.0
12-18	41	0	3	100	98	2.0
18-24	37	0	4	100	98	2.0
24-30	30	1	6	97	95	3.5
30-36	25	0	5	100	95	3.5
36-42	21	1	3	96	91	5.4
42-48	15	0	6	100	91	5.4
48-54	12	2	1	88	79	9.2
54-60	11	0	1	100	79	9.2

Table IV, B. Cumulative graft patency data for multiple vein segment bypass grafts undergoing revision

Interval (mo)	*No. of grafts at risk*	*No. of failed grafts*	*No. of grafts withdrawn*	*Interval patency (%)*	*Cumulative patency (%)*	*Standard error (%)*
0-3	18	0	0	100	100	0.0
3-6	16	0	2	100	100	0.0
6-12	15	1	0	94	94	6
12-18	12	0	3	100	94	6
18-24	9	0	3	100	94	6
24-30	6	1	2	89	83	10
30-36	3	2	1	73	56	15
36-42	2	0	1	100	56	17

36% by midgraft lesions that were not specifically related to technical errors. Mills[13] prospectively studied 227 infrainguinal reversed vein grafts over a 5-year period and found 33 intrinsic graft stenoses in 29 compromised grafts, of which the majority (53%) were juxtaanastomotic, and only 29% of the lesions were in the middle portion of the graft. In 1991 Bandyk et al.[14] reported his experience with vein graft revision in a series of 83 infrainguinal saphenous bypass grafts, 94% of which were in situ reconstructions. The most common cause of graft failure was intrinsic graft stenosis, which usually developed within the first year after surgery. The distribution of stenoses in this predominantly nonreversed saphenous graft series was 31% juxtaanastomotic, 45% midgraft, and 24% native artery progression. Bandyk also reported a 21% restenosis rate after VPA and concluded that interposition grafting may be a more durable option. In that report, however, VPA was used for only 31 revisions and 15 were at an anasto-

Table V, A. Cumulative patency data for lower extremity autogenous bypass grafts undergoing revision for midgraft anastomotic stenoses

Interval (mo)	*No. of grafts at risk*	*No. of failed grafts*	*No. of grafts withdrawn*	*Interval patency (%)*	*Cumulative patency rate (%)*	*Standard error (%)*
0-3	41	0	0	100	100	0
3-6	39	0	2	100	100	0
6-12	35	1	3	97	97	2.7
12-18	31	0	4	100	97	2.7
18-24	27	0	4	100	97	2.7
24-30	20	1	6	96	93	4.7
30-36	15	1	4	96	89	6.4
36-42	13	0	2	100	89	6.4
42-48	8	0	5	100	89	6.4
48-54	6	1	1	88	77	11
54-60	5	0	1	100	77	11

Table V, B. Cumulative patency data for lower extremity autogenous bypass grafts undergoing revision for distal anastomotic stenoses

Interval (mo)	*No. of grafts at risk*	*No. of failed grafts*	*No. of grafts withdrawn*	*Interval patency (%)*	*Cumulative patency (%)*	*Standard error (%)*
0-3	20	1	0	100	95	4.9
3-6	18	0	1	100	95	4.9
6-12	17	0	1	97	95	4.9
12-18	16	0	1	100	95	4.9
18-24	14	0	2	100	95	4.9
24-30	12	1	6	96	88	8
30-36	10	1	1	96	81	10
36-42	7	1	2	100	71	13
42-48	4	0	3	100	71	13

Table VI. Cumulative patency data for lower extremity autogenous bypass grafts revised with vein patch angioplasty

Interval (mo)	*No. of grafts at risk*	*No. of failed grafts*	*No. of grafts withdrawn*	*Interval patency rate (%)*	*Cumulative patency rate (%)*	*Standard error (%)*
0-3	35	1	0	97	97	2.8
3-6	33	0	1	100	97	2.8
6-12	29	1	3	97	94	4.1
12-18	26	0	3	100	94	4.1
18-24	22	0	4	100	94	4.1
24-30	17	0	5	97	94	4.1
30-36	16	0	1	100	94	4.1
36-42	13	1	2	93	87	7.5
42-48	9	0	4	100	87	7.5
48-54	6	0	3	100	87	7.5
54-60	5	0	1	100	87	7.5

motic stenosis. In addition, jump or sequential grafts had a high failure rate (52%), although the majority of these revisions were to tibial vessels at the ankle level.

In our study of 95 operations to correct 112 anatomic stenoses in 67 infrainguinal autogenous reconstructions, we have demonstrated that an aggressive surgical approach to the failing or failed vein graft can be rewarded with durable results, although higher-risk composite grafts are generally at a disadvantage from the outset. Our philosophy of vein graft revision has generally been to correct simple lesions simply, favoring an approach that conserves conduit when possible without compromising the quality of the operation. It has been our policy to construct the

original bypass grafts and leave the graft in the subcutaneous position (94% of grafts reported) to facilitate vein graft surveillance and repair. In addition, we favor the early use of postoperative anticoagulation therapy (used in 89% of secondary revisions), which may account for the fact that only eight of 37 grafts in patients who had rest pain or tissue loss were occluded. VPA was the most frequently performed revision operation, comprising 53% of the repairs in this series. Focal stenoses that were amenable to VPA comprised 71% of the midgraft lesions observed, compared with 27% that were more diffuse and required jump or interposition grafts. These midgraft lesions were predominantly neointimal hyperplastic in nature and appeared within 6 to 18 months, in contrast to the atherosclerotic midgraft stenoses (7%) that appeared later (Fig. 1). Vein grafts that underwent VPA as the sole method of revision during the life of the graft had an 87% CGP rate. We observed only 6 recurrences after 60 VPAs, for a 10% recurrence rate. Two recurrences were successfully treated with a second vein patch, whereas a third stenosis recurred a second time and went on to undergo and interposition grafting procedure. Stenosis in one distal anastomotic vein patch recurred and was corrected with a distal jump graft. When life-table analysis was applied to grafts that underwent VPA compared with those that underwent interposition grafting procedures, no significant difference in the CGP rate was observed (Fig. 4). Although the graft lesions that were treated with VPA rather than interposition grafts were more focal in nature than the more diffuse hyperplasia in the latter; grafts revised with either method had reasonable patency rates in line with previous reports.[7,9,13-16] Jump grafts had an 83% CGP rate when they were used predominantly for distal anastomotic stenoses, in spite of the fact that six of the 25 procedures were performed for associated progressive distal atherosclerotic disease. Although Bandyk[14] has reported a high failure rate for jump grafts constructed from cephalic vein, nine of the 25 jump grafts in this review were single-segment or composite arm vein reconstructions, usually of median basilic vein. Of these nine grafts, two failed within 24 months, one required a subsequent vein patch, and four have remained patent beyond 30 months. We have increasingly favored the use of jump grafts over VPA for distal anastomotic stenoses because the exposure of the distal target artery through unscarred tissue planes is technically simpler and less morbid, especially at the tibial level. Obviously this strategy depends on conduit availability as well as the distal stenosis level.

This study reaffirms the value of regular clinical and noninvasive laboratory follow-up of lower extremity vein grafts, as 25% of the limbs studied were completely asymptomatic at the time of threatened vein graft revision. Our policy with respect to vein graft stenosis has been an aggressive one, tending toward early revision if the patient is medically fit and has autogenous tissue available. This study demonstrates that a revision strategy that favors VPA for focal midgraft stenoses, vein interposition for diffuse midgraft stenoses, and jump grafts for distal anastomotic and native artery stenoses can provide acceptable graft patency and limb salvage rates with reasonable morbidity and mortality rates (Table VI).

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DISCUSSION

Dr. Daniel B. Walsh (Lebanon, N.H.). We have just heard an analysis of the 5-year experience in graft salvage of a thoughtful group of vascular surgeons. They are seeking the most appropriate operation to prevent or overcome graft failure. This retrospective analysis examines the experience of 58 patients who underwent 95 operations to correct 112 anatomic lesions. The patients and the lesions compared are an issue. Are the small groups comparable? Is there enough material for statistical power? Is lesion behavior independent of patient characteristics? As with all our experiences, the problem is separating fact from variation.

Clearly this study demonstrates again the necessity for regular quantitative follow-up of lower extremity vein grafts. This study also confirms what Dr. Dennis Bandyk has taught us: revise vein grafts before they fail and your long-term limb salvage and graft patency rates will improve. There is a cost, however. The perioperative morbidity rate is 9%, and hemorrhagic complications occur in 3% of patients.

The authors have adopted a strategy of repair of focal lesions. My own experience agrees. Repair of defects and failing vein grafts is appropriate. I worry, however, that sacrificing one long piece of vein by periodic resections for interposition grafts and angioplasty procedures after lysis of a failed graft does not yield the longevity of graft patency that is provided by total graft replacement with an available venous conduit of suitable length.

My question to the authors is, when a suitable length of replacement graft is available and a bypass graft has failed, do they prefer whole-graft replacement or would they opt for lysis or thrombectomy and replacement of a segmental lesion? For me, this remains a most difficult clinical question. I would be eager to hear the authors' opinion.

Dr. Theodore R. Sullivan, Jr. I think that the answer to the question of whether to perform whole-graft replacement versus revision obviously depends on a number of issues. First, is the graft thrombosed or is it patent but on the verge of failure? That's clearly a big distinction. Dr. Whittemore and other investigators have shown that graft salvage after thrombectomy of a thrombosed graft is markedly reduced. Dr. Belkin from the same institution has subsequently shown that opening a thrombosed graft with lytic therapy also has inferior patency compared with failing grafts that are revised before thrombosis. However, we do use these maneuvers when we have patients whose grafts have failed and we do not have an available conduit for a replacement bypass. This issue of conduit availability does come up fairly frequently, in that 38% of the patients in this study had had previous saphenectomy for a number of reasons. In addition, because we are a tertiary referral center, patients frequently come to us having had numerous bypass attempts or failures and have no greater saphenous vein available. In those instances, we resort to arterial reconstruction with arm vein and lesser saphenous vein, and rarely polytetrafluoroethylene.

If the patient has had a failed vein graft that is thrombosed and there is an available saphenous vein in the other leg, we will definitely use it, provided that the other leg isn't critically ischemic as well. That is sometimes a very difficult call to make. On the other hand, if we have a patient who has a hemodynamically significant focal stenosis, that is a relatively simple problem to fix with a vein patch or a limited interposition graft. We would obviously perform the simple repair first using arm vein or a saphenous vein branch before sacrificing the entire contralateral saphenous vein. So it really depends on the extent of the lesion, and whether the graft is thrombosed.

Dr. Frank J. Veith (Bronx, N.Y.). Did you separate the patients whose grafts had lytic therapy and then underwent a revision? Because your patency rates overall are so good, if you had a large number of these lysed revisions then your results would differ greatly from what most of us observe, that is, that these are not very favorable grafts and do not do well in the long term. So did you separate these lysed revised cases and analyze them separately?

Dr. Sullivan. We had 11 grafts that were thrombosed at the time of presentation, and nine of those underwent lytic therapy. When we split those out, we had an insufficient graft number to obtain statistical significance, but anecdotally, the patency results were actually pretty good. Six of the nine grafts that had failed and then underwent lytic therapy were still open at the last follow-up, and two of these were open as far out as 45 months. So we are encouraged by this, but I did not report it because the results are premature.

Dr. Dominic A. DeLaurentis (Philadelphia, Pa.). Could you tell us the size of the patient pool during that 5-year period? How many infrainguinal bypass procedures were performed? And could you give us the breakdown

between in situ and reversed saphenous vein bypass procedures. I realize that most of your redos here were with in situ grafts, but could you give us those two figures?

Dr. Sullivan. I will clarify that. Ninety-four percent of the original bypass graft procedures were reconstructions with nonreversed saphenous vein. They were evenly split between in situ bypass grafts and nonreversed translocated grafts. Reversed saphenous vein bypass grafts only comprised 6% of the grafts in this series, so comparison of these two groups is not possible.

I cannot provide you with an accurate incidence of graft revision, because to do that I would need a reliable denominator. This group of patients who underwent vein graft revision over a 5-year period was selected from a series of infrainguinal reconstructions that were actually performed over an 8-year period that both preceded and overlapped the study period. In addition, 15% of those patients were referred from outside hospitals with failing or failed grafts to be revised. Furthermore, a small percentage of the patients who developed failing vein grafts did not undergo revision of the graft, whether it be for medical or conduit-availability reasons, and are not included in our series. So I cannot give you an accurate denominator to provide a reliable revision rate, although of the original bypass procedures performed at our institution, the revision rate is 8%.

Dr. Thomas S. Riles (New York, N.Y.). I certainly would go along with the idea that these stenoses should be surgically repaired when identified. I'm a little curious, in the room here with a lot of people who are very enthusiastic about endovascular surgery and stents and so forth, that the topic hasn't been brought up as to what role these techniques would play in the treatment of midgraft lesions. What is your thinking on that topic? Perhaps some of the others present would like to discuss that, as well.

Dr. Sullivan. Dr. Sanchez and Dr. Veith have reported data from their institution with favorable results using balloon angioplasty on failing lower extremity bypass grafts of all sorts, polytetrafluoroethylene included. The interesting thing about that series was that the majority of the lesions on which they were performing balloon angioplasty were inflow lesions or lesions near the proximal anastomosis. Dr. Berkowitz subsequently published an article on reversed bypass grafts, and again there was a predominance of lesions in the proximal juxtaanastomotic area, and they used balloon angioplasty and had very good results.

We and our angiographers have been less than enthusiastic about performing balloon angioplasty in this setting because the majority of the stenoses that we discover are in the middle portion of the graft or at the distal anastomosis. It seems logical that these lesions are not going to do as well with balloon angioplasty as the proximal native artery or proximal anastomosis lesions. In this series, we only had six balloon angioplasty procedures total, and five of those were performed in conjunction with other surgical procedures. So we will use angioplasty selectively for proximal lesions, but as far as midgraft stenoses are concerned, we favor surgical revision. We think that this is a relatively simple procedure to perform and it has durable patency.

Dr. Henry D. Berkowitz (Philadelphia, Pa.). I rise only to comment on performing balloon angioplasty on vein graft stenoses, which has achieved only modest success in most studies of graft revision. I noticed that you analyzed the patency data of intact saphenous grafts separately from that of alternative vein grafts. I recently did the same analysis with a large series of angioplasty-treated graft stenoses. The cumulative 5-year patency rate of the saphenous grafts was 80%, compared with 42% for the alternative vein grafts. Furthermore, most of the poor results occurred in grafts that had strictures at the site of vein-vein anastomoses. Careful analysis of data, such as you have done, that identifies the subgroups of graft strictures that can be successfully treated without surgery is a valuable contribution.

Dr. Peter J. Pappas (Newark, N.J.). My understanding of performing angioplasty in arteries is that the reason why it works is because there is a controlled dissection that is created, whereas in a vein graft that has intimal hyperplasia, you get recoil when you dilate the artery because of the elastic properties of the intimal hyperplastic segment, and that's why balloon angioplasty doesn't work. I think the issue is whether placing a small coronary-type stent such as is often done when performing percutaneous transluminal angioplasty with the coronary circulation would be of any benefit, and I am not aware of any literature or any studies that have looked at that, are you?

Dr. Sullivan. I am not aware of any data on this issue, but I agree with you that after angioplasty of a hyperplastic vein graft stenosis, there is going to be a significant recoil effect. However, as soon as you introduce a stent into an already diseased vein graft, you are going to get a fairly vigorous intimal hyperplastic response. When you take a relatively small-diameter vein graft and add to it the hyperplastic reaction around a stent, that is going to compromise the lumen diameter. Placing a stent across a focal stenosis in a sense burns a bridge for a simple surgical revision with a vein patch.

Dr. Pappas. I was also intrigued when you said that there was a 10% incidence of restenosis in your vein patches. I have similarly started to find that in the vein patch group, I have started to see restenoses as well, and I have noticed that some of the other surgeons around the country have advocated using interposition grafts rather than vein patches. What is the policy at your institution, have you put in interposition grafts over vein patches at all?

Dr. Sullivan. We reserve interposition grafts for more diffuse lesions, not for the short focal stenoses that are amenable to a vein patch. The reason is that you need a reasonable caliber diameter vein to reconstruct an interposition graft. To create a vein patch, as long as the vein is reasonably healthy, you can use a very small-caliber vein. So when vein conservation is the issue, the interposition graft is not as good an option, and I think that we have shown that the vein patch is just as durable.

Dr. Michael A. Golden (Philadelphia, Pa.). I have two comments. The first is in reference to the reported suboptimal long-term performance of vein grafts after occlusion and thrombolysis. If a vein graft that has been lysed and found to have a focal lesion can be repaired, however, it may be possible to get an extra 2 to 5 years of graft patency out of the graft without the loss of much additional vein. Many patients do not have another yard of greater saphenous vein, and if they do have it, they may need it for a more diffuse problem with their bypass graft, where graft replacement is required, or for contralateral disease.

The second is in reference to question that was posed earlier in the discussion about placing stents in vein grafts. In saphenous vein aortocoronary bypass grafts there is an experience with balloon angioplasty and stenting. It is not extensive and does appear to be associated with some problems. However, in patients who have stenotic vein grafts after coronary artery bypass grafting procedures, angioplasty with stent deployment is a much less invasive procedure than repeat sternotomy and graft revision. In some instances good short-term results can be achieved. Stent placement is associated with stimulation of intimal hyperplasia and would likely not yield excellent long-term results.